Chapter 7

Diseases of the Lower Respiratory Tract and Thorax

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I. DISEASES OF THE LOWER RESPIRATORY TRACT

A. Pneumonia in young animals

1. Pneumonia in neonates. Clinical signs of pneumonia in all species generally include fever, tachypnea or dyspnea, anorexia, and, particularly with bacterial involvement, depression. Accompanying these signs are a productive cough and a bilateral mucoid to mucopurulent nasal discharge. On chest auscultation, loud bronchial tones over the trachea and lungs are audible, with adventitious sounds (wheezes, crackles) over the lung fields.

2. Pneumonia in foals. Respiratory disease of foals can have a variety of causes, both infectious and noninfectious. Of these causes, the more important infectious diseases are discussed in this chapter.

a. Overview

(1) Viral pneumonia

(a) Patient profile and etiology. The viral agents that result in signs of pneumonia in foals include the equine herpesviruses (EHV-1, EHV-4), influenza virus, and adenovirus. Pure viral pneumonia in foals is associated with immunocompromised, debilitated foals (e.g., failure of passive transfer, combined immunodeficiency, steroid therapy, poorly nourished foals). The main problem with these infections is the potential to predispose to more clinically serious secondary bacterial pneumonia.

(b) Clinical findings are similar to those described for neonatal pneumonia because they are seldom specific for any one viral agent. In foals, the severity of pneumonia is often poorly correlated to chest auscultation findings. For the pathogenesis, diagnostic plans, and therapeutic plans for these agents, refer to Chapter 6.

(2) Bacterial pneumonia

(a) Patient profile and etiology. A variety of bacteria can be associated with pneumonia in foals.

(i) Streptococcus ***zooepidemicus*** is the most common [see I A 2 b (1)].

(ii) ***Actinobacillus equuli*** can cause pneumonia in older foals, whereas this organism causes septicemia and nephritis in very young foals.

(iii) ***Klebsiella pneumoniae*** causes a severe pneumonia in septicemic foals, as do the Salmonella species in foals 2–3 weeks of age, in which other signs include diarrhea and arthritis.

(iv) ***Escherichia coli*** can be a cause of embolic pneumonia secondary to septicemia.

(v) ***Bordetella bronchiseptica*** has been a cause of bacterial pneumonia in foals, though it is usually not a primary pathogen.

(vi) ***Rhodococcus equi*** is a specific pathogen for foals, with lung abscessation as the primary lesion [see I A 2 b (2)].

(b) Risk factors

(i) In the early stages of life, foals with septicemia usually have an associated bacterial pneumonia. Clearly, the elements that predispose to septicemia (e.g., problems with sufficient passive transfer) are key factor.

(ii) Older foals are at risk to secondary bacterial pneumonia when they experience respiratory viral infection. In these cases, there is usually stress of transport and overcrowding, as may occur accompanying transport of the mare to be bred.

(iii) Inadequate ventilation (excessive dust levels, poorly cleaned stalls), poor nutrition, and parasite migration are thought to contribute to foals being predisposed to developing pneumonia, although these are not sufficient causes on their own.

b. Specific conditions

(1) Streptococcus ***zooepidemicus*** pneumonia

(a) Patient profile and history is variable because the organism is an opportunistic pathogen in horses of all ages and ubiquitous in their environment.

(b) Clinical **findings.** Signs include tachypnea, anorexia, depression, fever, abnormal lung sounds, and possibly a cough. None of these signs are specific for S. *zooepidemicus* infection. However, this organism can cause lymph node abscessation, in contrast to the other opportunistic bacteria, and could resemble *S.* equi infection in the early stages before signs of pulmonary involvement.

(c) Etiology and pathogenesis

(i) Etiology. *S.* zooepidemicus is a normal inhabitant of the equine upper respiratory tract and does not normally invade intact mucous membranes. Any of several stressors (e.g., poor air quality, inadequate ventilation, damage to respiratory epithelium by viral infections) to the normal respiratory defenses may predispose this organism to spread to the lungs.

(ii) Pathogenesis. Acute infections result in severe fibrinopurulent bronchopneumonia with hemorrhage, whereas less fulminant infections cause abscesses in the lungs, lymph nodes, and occasionally in the pleura (pyogranulomatous pneumonia).

(d) Diagnostic plan and **laboratory** tests

(i) A transtracheal aspirate aids diagnosis and rapidly directs specific treatment. Typically, there are gram-positive cocci noted in the fluid recovered, which is evidence of Streptococcus involvement. Thus, while awaiting culture of the organism, treatment can then be selected against this family of organism.

(ii) Thoracic radiographs (used for foals only because these are not usually available for adult horses) can confirm the presence of pneumonia and may detect lung abscesses, which require far more prolonged **treatment.**

(iii) Hematology usually reveals leukocytosis and increased fibrinogen, but these changes are not specific for this infection.

(e) Therapeutic plan

(i) Penicillin is the drug of choice for S. *zooepidemicus* infections. If the clinical signs are marked (indicating fulminant infection), foals should be started on intravenous medications, such as sodium or potassium penicillin (20,000 IU/kg intravenously every 6 hours). For less severe clinical signs, affected foals (and adult horses) respond well to **procaine** penicillin (20,000 IU/kg intramuscularly every 12 hours).

(ii) An alternative treatment is **trimethoprim-sulphadiazine** (30 mg/kg orally every 12 hours). Treatment should continue well beyond resolution of clinical signs. Treatment effectiveness should be monitored using blood work and serial chest radiographs.

(f) Prevention. Because there are 13 serotypes of S. *zooepidemicus,* there is no vaccine available. Therefore, preventive measures include attention to hygiene and reduction of stress, particularly in the neonatal period. For older foals and even adult horses, adequate rest and a stress-free environment following respiratory viral infection should be ensured for at least several weeks.

(2) ***Rhodococcus*** equi pneumonia

(a) Patient profile and history. This is an infectious respiratory disease of foals usually 4 months or younger that is usually sporadic in occurrence on any one farm. R. equi is an opportunistic bacterium that appears to infect the foal when maternal antibody levels are waning and viral infections impair defense mechanisms. Affected foals can have a prolonged course of chronic respiratory signs or exhibit a sudden onset of severe respiratory distress that can be rapidly fatal.

(b) Epidemiology

(i) Foals between the ages of 1 and 3 months are most commonly affected, but the onset of signs can range from age 2 weeks to 6 months. The disease is rare in adult horses and occurs only in immunocompromised animals.

(ii) There appears to be an increased incidence during the hot, dry months of summer, which may be attributable to the increased spread of soil- and fecal-born organisms in dusty conditions. During these dry conditions, the risk of infection can be reduced if the foals are put onto grass pasture. Morbidity is low, but the case fatality rate is high if left untreated.

(c) Clinical findings. The signs of disease appear to vary with the age at which foals become affected.

(i) Young foals affected after 1 month of age often have acute signs of illness with fever, respiratory distress, anorexia, and, in some cases, swollen joints.

(ii) Older foals can develop serious lesions in the absence of marked clinical abnormalities, which then begin to manifest as a persistent cough and a progressive increase in respiratory effort at rest, with crackles and wheezes audible over the chest. These foals often suckle normally and have no fever during such disease progression, but they become emaciated. In some foals, diarrhea may follow or accompany the respiratory signs, but nasal discharge and lymph node enlargement in the throat region are absent.

(iii) Other clinical signs can include arthritis in one or several joints and uveitis, both presumed to be immune mediated. In foals with these signs, the slow development of apparent pneumonia masks the severity of underlying pulmonary disease; therefore, it is prudent to suspect R. equi infection in a foal with nonresponsive pneumonia.

(d) Diagnostic plan and laboratory tests. Although clinical signs are usually suggestive of pneumonia, it is important to establish an etiologic diagnosis in the case of R. equi infection for appropriate treatment and advice on prognosis. Transtracheal aspirate cytology and culture, along with thoracic radiography, are the most valuable diagnostic tests.

(i) On transtracheal wash, there are usually large numbers of neutrophils, and the bacteria (often intracellular) can have a characteristic "Chinese letter" appearance. Culture of the sample should be positive for R. equi, but in those foals that have been treated with antibiotics, there may be no bacterial growth.

(ii) Radiography. Classically, "cotton-puff abscesses" of the lung are present on chest radiographs, although the radiographic image can also consist of diffuse pulmonary infiltration and air bronchograms with hilar lymphadenopathy.

(iii) Blood work. A complete blood cell count (CBC) and fibrinogen level, although not definitive for *R. equi,* are useful for monitoring the course of the inflammatory process. Affected foals usually show a neutrophilic leukocytosis with hyperfibrinogenemia, accompanied by anemia of chronic disease. The fibrinogen response can also be used as a guideline for response to treatment.

(iv) Serologic tests have been of limited value because although foals with pulmonary infection caused by R. equi show seroconversion to this organism, normal foals in the first few months of life can also show seroconversion as a result of intestinal colonization by the organism.

(e) Etiology and pathogenesis

(i) Etiology. R. equi is a gram-positive pleomorphic rod isolated from the soil and feces of normal horses. Although it causes disease in horses, it also can cause cervical abscesses in swine and abscesses in the intestinal and pulmonary lymph nodes of cattle. Because of the ubiquitous nature of the organism, most foals are exposed to infection, but only a few develop disease. The organisms can survive in moist soil for periods longer than 1 year. In some farms where R. equi pneumonia appears endemic with multiple cases each year, there is likely a bacterial load in the environment.

(ii) The route of infection is not definitively known, but most evidence supports aerosolization or inhalation of soil-derived bacteria. An alternate route suggested is ingestion and intestinal colonization with subsequent hematogenous spread.

(iii) Pathogenesis. When the bacteria reach the lung, they induce a suppurative pyogranulomatous bronchopneumonia with characteristic abscessation. Abscessation occurs because R. equi are able to survive within macrophages. This ability to live and multiply within phagocytes results from failure of phagosome–lysosome fusion and also from the lack of a superoxide anion response in the equine pulmonary macrophage following ingestion of *R.* equi.

(iv) Parasite migration through lungs may contribute to development of disease. Other disease manifestations include nonseptic arthritis, ulcerative colitis, hepatic and splenic abscesses, vertebral abscesses, and uveitis,

(f) Therapeutic plan. Because the underlying process is a pyogranulomatous reaction, attributes of the selected antibacterial treatment should include good distribution and activity in lungs, adequate penetration into thick caseous abscesses, and penetration into cells to act on bacteria within macrophages and neutrophils.

(i) Rifampin (5–10 mg/kg orally every 12 hours) with erythromycin **estolate** (25 mg/kg orally every 6 hours) is the combination of choice. The erythromycin may cause a transient diarrhea but is usually self-limiting and abates on temporary withdrawal of the drug.

(ii) Treatment should be continued for at least 30 days. The fibrinogen concentration can be used as a guide for efficacy, a reduction signaling treatment effectiveness. Follow-up chest radiographs can be helpful in determining the response to treatment.

(iii) If foals have periodic serum biochemical tests during treatment, enzyme elevations suggestive of cholestasis may occur but can be expected with use of these drugs.

(g) Prevention. Because the organism is ubiquitous in the foals' environment, it is difficult to control.

(i) Minimizing exposure. With the increased risk associated with dusty environments, exposure can be minimized by maintaining the mare and foal on grassy **paddocks** or pasture. The pasture management technique of routine disposal of feces may also decrease exposure.

(ii) Administration of hyperimmune serum. As yet, there is no effective vaccine for use against this disease. However, recent studies indicate that administering hyperimmune serum, obtained from mares that were given an autogenous vaccine, to foals at risk did limit the severity of disease produced by experimental challenge. Additionally, field studies have shown that administration of hyperimmune serum to foals in their first month of life has resulted in a significant reduction in the disease incidence. Unfortunately, vaccination of the dam to boost the specific colostrally transferred passive immunity has not been met with similar positive results.

(3) ***Pneumocystis carinii*** pneumonia. *P.* carinii is an ubiquitous sporozoan that causes interstitial pneumonia in immunocompromised humans. This opportunistic pathogen is associated invariably with other organisms and has occurred in foals with R. equi, in immunocompromised foals (e.g., combined immunodeficiency), or in foals taking corticosteroids on a long-term basis. Although clinical signs reflect a bacterial bronchopneumonia, this disease is usually diagnosed postmortem.

3. **Enzootic** pneumonia in calves

a. Patient profile and history. This disease occurs almost exclusively in calves raised indoors. Therefore, enzootic pneumonia is found mainly in dairy herds. Although this disorder can occur as early as the first week of life, this disease is most common in calves between 2 and 5 months and up to 1 year of age. Fall and winter are the times it is most often observed. Poor air quality is a contributing factor to infection.

b. Clinical finding

(1) Affected calves in the acute stage have a moderate fever (40°C–40.5°C) and a harsh, hacking cough that is easily induced by pinching the trachea.

(2) Tachypnea and dyspnea are often present, with increased bronchial tones audible over the cranial lung fields suggestive of lung consolidation. Crackles and wheezes at the periphery of areas of consolidation may also be audible, suggesting bronchiolitis.

(3) Calves are usually alert unless there is a significant bacterial component to the pneumonia, in which case the fever may be higher.

c. Etiology and pathogenesis. At approximately 2 months of age, calves' immunity to respiratory infection (a combination of waning colostral immunity and the slow development of an independent response) is at its lowest point. This may be a key reason that most of the calf pneumonia problems caused by infections begin to appear at this age.

(1) Infection may begin as a viral respiratory infection, which may resolve or become complicated by a variety of bacteria, mycoplasmal organisms, or both (multifactorial).

(a) Viral agents include parainfluenza-3 (PI-3), bovine respiratory syncytial virus (BRSV), and bovine viral diarrhea (BVD), of which PI-3 and BRSV appear the most significant. BRSV is increasingly implicated as a major factor in the genesis of enzootic pneumonia. Unless there is secondary complication by bacteria, the disease is generally an interstitial pneumonia affecting the cranial lung lobes. Other viruses of cattle (e.g., rhinoviruses, adenoviruses, reoviruses) are not considered important

(b) Mycoplasmal organisms, including Mycoplasma *bovis,* M. dispar, M. *bovirhinis,* and *Ureaplasma,* also are found in many cases of enzootic pneumonia.

(i) These organisms inhabit bronchiolar epithelium; thus, they cause ciliary destruction and changes in mucus composition as goblet cells proliferate and mucus hypersecretion occurs.

(ii) The classic pathology attributed to mycoplasmal infection in these calves is peribronchial lymphocytic cuffing of the bronchioles. It appears that the adult cattle act as mycoplasma carriers and transfer the organisms by aerosol up to several meters' distance to calves.

(c) Bacterial **agents** involved in this pneumonia include many of the organisms that can be found in the pharyngeal region of normal calves, including *Pasteurella* haemolytica, *P. multocida,* and *Actinomyces pyogenes.* Chlamydia species also have been involved in some mixed infections. Involvement of these secondary agents results in purulent secretions accumulating in the airways and eventually the formation of microabscesses, bronchiectasis, or both. The pathology that occurs is a consolidation of cranial, middle, and accessory lobes with bronchiolitis and alveolitis, with varying degrees of consolidation and suppuration depending on the numbers and type of bacterial involvement.

(2) Environmental factors

(a) The infectious agents are seldom able to induce pneumonia in calves with-

out the added stress of poor or absent ventilation in the housing areas for the calves.

(b) Colder weather often precipitates problems when the producers try to prevent the calf housing temperature from dropping by reducing fresh air circulation. Consequently, there is increased humidity in the ambient air of the calves, which allows pathogen multiplication. This is compounded by a high pathogen density developing with the lack of fresh air to dilute the ambient air.

(c) Another factor of adverse air quality is the increased exposure to noxious gases (e.g., ammonia) that can adversely affect mucociliary clearance.

(3) Management factors include the crowding and mixing of various age groups that frequently occurs with indoor housing, particularly in older barns.

d. Diagnostic plan and laboratory tests

(1) Clinical signs of pneumonia are the main mode of diagnosis. Attempts can be made at virus isolation or viral serology, but these efforts are often unrewarding and unlikely to change the methods of management of the problem.

(2) Laboratory studies. For the individual calf, a transtracheal aspirate can be performed. Cytology findings of increased numbers of degenerate neutrophils with numerous intracellular bacteria implicate secondary bacterial involvement. In these cases, bacterial culture and sensitivity may aid in guiding antibiotic treatment of the calf. A CBC indicates changes of leucocytosis and hyperfibrinogenemia where bacteria are involved.

(3) Radiography. For calves that have a prolonged course of pneumonia and respond poorly to antibiotics, thoracic radiographs may identify processes such as abscessation or bronchiectasis, indicating a poor prognosis.

e. Therapeutic plan. Acutely affected calves often respond well to antibiotics, unless the infection is purely viral, as can occur in some BRSV infections.

(1) Although many of the commonly available antibiotics for cattle can be used, drugs that are effective against the mycoplasmas, such as tylosin tartrate (10 mg/kg intramuscularly daily for 3–4 days) or oxytetracycline (10 mg/kg intramuscularly daily for 3–4 days), provide appropriate coverage against the spectrum of possible organisms involved.

(2) More recently, **flunixin** meglumine (2.2 mg/kg intravenously every 24 hours for 2–3 days) also has been advocated as an adjunct treatment because it may reduce lung inflammation in these calves.

f. Prognosis. In chronically debilitated calves that may have extensive lung damage, including abscessation, the prognosis for full recovery is poor, and therapy may not be cost-effective.

g. Prevention

(1) Vaccines. The commonly available vaccines against the viral pathogens are of questionable value, as colostral immunity usually interferes with adequate response at the time of administration.

(2) Management strategies

(a) For producers with major problems, individual outdoor calf hutches are highly recommended because they provide optimal ventilation and a level of isolation from older cattle.

(b) Producers with less severe problems can reduce the incidence of enzootic pneumonia with attention to adequate ventilation in the calf barn, which removes pathogens, noxious gases, and excess moisture.

(c) Adequate colostral feeding also may prevent enzootic pneumonia because survival of calves with respiratory disease has been directly correlated with serum immunoglobulin G levels.

4. Pneumonia in lambs and kids is similar to that in adult sheep and goats (see I B 3).

5. Pneumonia in piglets

a. Etiology. Pneumonia in piglets can be caused by viruses, including pseudorabies, swine influenza (see Chapter 6), and the recently described porcine reproductive and respiratory syndrome (PRRS).

(1) PRRS, also known as porcine endemic abortion and respiratory syndrome, blue-eared pig disease, swine infertility and respiratory syndrome, and originally "mystery disease of swine," was first recorded in the United States in 1987 and is thought to be caused by the Lelystad virus in Europe and a similar, if not identical virus, in North America. Since being reported in the United States, this virus has been recorded in Canada and several countries in Europe.

(2) Transmission appears to be airborne because even well-managed and isolated herds can become infected.

b. Clinical findings

(1) Adults. Signs include anorexia and fever lasting several days, after which there may be mid- to late-term abortions with large mummified fetuses or partially autolysed fetuses, increased percentages of stillbirths, and weak piglets. This period of reproductive problems is followed or accompanied by severe respiratory disease in nursing or weaned piglets.

(2) Piglets. Signs include dyspnea, polypnea, and decreased growth. The abdomen, ears, or vulva may appear bluish (hence the name blue-eared pigs). In a litter, up to 50% have anorexia, 30% show respiratory distress, 10% have fever, and only 5% show cyanosis. Morbidity in weanling pigs can be as high as 30%, with mortality of 5%–10%.

c. Diagnostic plan and laboratory tests. There are several serologic tests available to detect seroconversion to the virus, but because of the possible widespread infection, the serodiagnosis should be based on a herd rather than on a sample from an individual animal. There are no characteristic lesions in the aborted or stillborn piglets or in the sows. Piglets will have a proliferative and necrotizing pneumonia and, in some cases, a purulent bronchopneumonia.

d. Differential diagnoses. This disease needs to be differentiated from other conditions that cause abortion, stillbirths, and weak piglets, such as pseudorabies, parvovirus, leptospirosis, and the encephalomyocarditis or hog cholera viruses.

e. Therapeutic plan and prevention. Because there is no specific treatment available, control is the main focus. There is not sufficient data available for understanding the methods of transmission, but restricting the movement of pigs from affected to unaffected areas has slowed the spread.

B. Pneumonia in growing and mature animals

1. Equine bacterial pneumonia

a. Patient profile and etiology. Pneumonia in the adult horse is not common and is usually secondary to upper respiratory virus infection [see I A 2 b (1)] or occurs after stressful events (e.g., shipping), in which case it often develops into pleuritis (see I E).

b. Diagnostic and therapeutic plans. The guidelines given for foals are used, with the exception that chest radiographs are not obtained because they are not readily available for monitoring progress in the adult horse.

2. Bovine respiratory disease complex (shipping fever, pneumonic pasteurellosis)

a. Patient profile and history. This disease can affect cattle of any age, breed, or sex. However, because of current management practices, this condition occurs primarily in young cattle between the ages of 6 and 18 months. Characteristically, the disorder follows a stressful event (e.g., shipping, mixing in sales), occurring 2–14 days later, and is one of the major causes of morbidity and mortality in feedlot cattle in North America.

b. Clinical findings

(1) Affected cattle are depressed, usually anorexic, and have rapid shallow respirations with a weak, productive cough. Early signs of the depression may be simply a failure to stretch freely when encouraged to rise. Fever is often as high as 41°C, and examination of the penmates may reveal fever in animals that otherwise appear clinically normal.

(2) In the early stages of the disease, lung sounds only consist of increased breath sounds. In the advanced stages, crackles and wheezes become readily audible, and there may be pleural friction rubs in some animals. A mucopurulent

nasal discharge, crusty nose, and ocular discharge may appear, along with a gaunt appearance to the abdomen after several days of not eating.

c. Etiology and pathogenesis

(1) Etiology

(a) This disease is a fibrinous pneumonia caused by Pasteurella ***haemolytica*** (biotype A, serotype I) and, less commonly, Pasteurella multocida or *Hemophilus* somnus. Experimentally, these organisms alone do not produce disease without massive doses or manipulation of the host with viral pathogens or severe stressors.

(b) In naturally occurring disease, the associated stressful events are numerous and include weaning, transportation, sudden changes in climatic conditions (e.g., temperature, humidity), inadequate or irregular feeding, water deprivation, crowding, dehorning, castration, and vaccination. Respiratory viral infection can be a factor, but in many outbreaks of naturally occurring disease, there is no preceding viral infection.

(2) Pathogenesis. P. haemolytica and P. multocida are normal inhabitants of the bovine upper respiratory tract. With the stressors, these organisms are able to colonize and proliferate in the lower respiratory tract. P. haemolytica stressors are associated with increased numbers and virulence of the strain found in the pharynx of cattle. Because this organism appears as the predominating agent in this disease syndrome, most investigation into pathogenesis has focused on P. haemolytica.

(a) P. *haemolytica* has four virulence **factors** that interact to produce disease: fimbriae, polysaccharide capsule, endotoxin, and leukotoxin.

(i) The bacterial fimbriae enhance the colonization and proliferation of bacteria in the upper respiratory tract.

(ii) The polysaccharide capsule inhibits complement-mediated serum killing as well as phagocytosis and intracellular killing of the organism.

(iii) Endotoxin is directly toxic to the bovine endothelium and greatly modifies cardiopulmonary hemodynamics. Also, it can alter bovine leukocyte functions.

(iv) Leukotoxin is a species-specific cytotoxin against ruminant leukocytes and platelets, acting as a pore-forming cytolysin.

(b) Transmission. Cattle either have the bacterial organisms in their nasopharynx or become infected by aerosol from other cattle. However, the disease does not appear to be contagious in the way it affects the individual.

(c) Route of **infection**

(i) With increased proliferation of the bacteria in the nasopharyngeal region and increases in virulence type, P. haemolytica is allowed to colonize the airways of the lung. The presence of P. haemolytica (lipopolysaccharide) attracts neutrophils to the lung as a normal defense mechanism.

(ii) Although local immunity such as immunoglobulin can be protective, some forms of systemic immunity can actually enhance intrapulmonary pathology of Pasteurella infections, possibly because of opsonization (immunoglobulin G) effect.

(iii) *P.* haernolytica produces an exotoxin during log phase growth that is cytotoxic for ruminant neutrophils and macrophages. Therefore, the increased bacterial phagocytosis induced by opsonization may merely cause increased cell death of the very cells (neutrophils and macrophages) that are intended for defense. Also, release or "spilling" of the cytotoxic contents of these cells can contribute to lung damage.

(iv) The pathology that results is a severe fibrinous pneumonia with increased capillary permeability, thrombosis, and coagulation necrosis. Bacterial cell death, which releases endotoxin (lipopolysaccharide), probably contributes to these changes. In contrast to earlier theory, viral involvement is not an essential component of this disease.

d. Diagnostic plan and laboratory tests

(1) Clinical findings of rapid onset of high fever, depression, and rapid, shallow, often guarded breathing, combined with a stressful event is usually sufficient for a tentative diagnosis.

(2) Laboratory analysis is seldom warranted with individual cases and is not diagnostic for shipping fever.

(a) Blood work. Predictably, there is a leukocytosis on the CBC, but there also can be leukopenia if endotoxemia is a major component. Hyperfibrinogenemia is usually present (greater than 7 g/L).

(b) A transtracheal aspirate yields septic, degenerate tracheal exudate containing gram-negative rods and often Pasteurella species on culture.

(c) If nasopharyngeal swabs are taken, *P.* haemolytica can be cultured, but there appears to be poor correlation of antimicrobial sensitivities between isolates from the nasopharynx versus the lung. Therefore, these samples should not be used to guide the choice of antibiotic for the treatment of acute cases.

e. Therapeutic plan

(1) Antimicrobial treatment. Early treatment of acutely affected cattle with almost any of the commonly available antimicrobials (e.g., oxytetracycline, trimethoprim-sulfadoxine, penicillin, sulfonamides) is highly effective.

(a) Surprisingly, a single-dose treatment in early stages is often sufficient, but severely affected cattle should be treated daily for 3–5 days.

(b) Delayed or irregular treatment, inappropriate dose, or premature termination of therapy in some cattle can result in increased mortality. Cattle with more than 50%–60% pulmonary consolidation generally respond poorly and frequently relapse. These animals may be individuals that were not detected in the acute stage or were not adequately treated early in the course of the disease.

(2) In addition to antimicrobial treatment, sick cattle should receive adequate shelter and good nutrition, particularly when the management system in place encourages crowding and competition for feed and water space.

(3) Response to treatment

(a) Antimicrobial-resistant strains of P. ***haemolytica*** have been appearing and, with resistance being plasmid mediated, may significantly increase the incidence of poor response to treatment.

(b) To assess adequate response to treatment, the rectal temperature should be used as a guide. A positive response is the abatement of fever within 48 hours of treatment

f. Prevention. Because this disease is so clearly linked to stressful events, a great deal of the prevention can be managed by avoiding or lessening the impact of known and preventable **stressors.**

(1) Preconditioning. The best example of stressful events is the practice of weaning calves simultaneous with castration, dehorning, and vaccination. By performing some of these procedures at a separate time, such massive stress can be lessened. This management method, called preconditioning or backgrounding, vaccinates and processes (castrates, dehorns, implants) calves before weaning, all of which should occur 3 weeks to 1 month before shipping. There are many variations of the timing and order of these preventive measures. However, the financial benefit to the producer is less clear, and unless a premium is paid for these calves at sale, the extra cost and effort may not be worthwhile.

(2) Vaccination against shipping fever has been a main focus of prevention but has not been particularly effective until recently.

(a) Where respiratory viral agents (e.g., IBR, PI-3, BRSV) are involved, the use of such antigens in vaccines may be beneficial when they are administered at least several weeks before weaning and shipping. Also, there has been evidence that vaccination with live products at the time of stress may increase morbidity or mortality.

(b) Progress has been made in creating vaccines against the pathogenic effects of P. ***haemolytica.*** Recently, a vaccine was produced to stimulate immunity against the exotoxin **or** leukotoxin that is produced as a major part of

the pathogenicity of P. haemolytica. This vaccine appears to protect against pulmonary disease in experimental models of disease but is less effective in field use. Current evidence from field trials suggests that this vaccine at best only marginally reduces the morbidity and mortality.

(3) **Chemoprophylaxis.** Another tact taken to reduce the morbidity and mortality of shipping fever is the administration of antibiotics at the time of stresses or upon arrival at a feedlot.

(a) Drugs used include long-acting oxytetracycline (LA200, 20 mg/kg) or tilmicosin (micotil, 10 mg/kg subcutaneously). Such treatment is presumed to be effective by postponing the bacterial invasion of the lung, so that the infection is usually less severe and less likely to result in relapses.

(b) Medication in feed or water has been used also, but both these approaches can give a false sense of security, reducing scrutiny of the cattle's health and leading to more advanced cases occurring before diagnosis. Also, with the current consumer concern regarding drug use in animals that are intended for human consumption, such practices can create problems for the image of the industry.

3. **Ovine** and **caprine** bacterial pneumonia

a. **Pasteurellosis**

(1) Patient profile **and** history. Outbreaks are often associated with sudden changes in environment or climate (e.g., when a flock of sheep is exposed to inclement weather shortly after shearing).

(2) Etiology and pathogenesis

(a) Etiology. As in cattle, *Pasteurella* haemolytica is the most common cause of bacterial infections of the respiratory tract in sheep and goats. However, the resulting diseases are not the same as those in cattle.

(i) There are two major **biotypes:** biotype A, which causes primarily pneumonia, and **biotype T,** which causes primarily acute septicemia in younger animals. Each biotype contains several serotypes.

(ii) P. *haemolytica* is commonly found in the nasal passages and tonsils of apparently healthy sheep. Several serotypes may be present at one time, but during outbreaks of respiratory pasteurellosis, one serotype usually predominates in both the upper and lower portions of the respiratory tract. The nasal carriage rate of P. haemolytica varies throughout the year and appears to increase as ewes approach parturition. Although P. haemolytica may occasionally cause mastitis in ewes, it is not often found in colostrum.

(b) Predisposing factors. Although P. haemolytica can be a primary pathogen in young lambs, older animals require predisposing factors for the occurrence of disease.

(i) In lambs, prior viral infection by such viruses as PI-3, followed in 4–7 davs with P. haemolytica infection can produce pneumonia that is similar to naturally occurring cases.

(ii) Adenovirus has also been incriminated as predisposing to naturally occurring and experimentally occurring pneumonia.

(iii) Other factors, such as exposure to inclement weather, transportation, and poor **nutrition,** are also predisposing factors.

(c) Pathogenesis. These factors combine to allow proliferation of the resident bacteria in the upper respiratory tract and invasion of the lung, where virulence factors, such as polysaccharide and leukotoxin (the specific cytotoxin against the ruminant leukocytes), cause extensive pulmonary damage.

(3) Clinical findings. Clinical disease is usually most severe in young lambs and kids, particularly with biotype A, which can cause a rapidly fatal septicemia.

(a) If detected **early,** affected lambs or kids appear dull and become prostrate before death in a matter of hours. Sick animals are often unobserved until the final stages of the disease.

(b) In the older animals, signs of respiratory disease are observed, with dyspnea, slight frothing at the mouth, cough, and nasal discharge. These signs are accentuated when the herd or flock is moved and affected animals fall behind the rest. Fever (greater than 40.5°C), depression, and anorexia also accompany these signs.

(4) Diagnostic plan and laboratory tests. Clinical signs of pneumonia are usually sufficient for a provisional diagnosis. There is little reported on the use of clinical pathology for assisting with a diagnosis. In outbreaks, there will likely be some mortalities on which **necropsy** can be performed.

(a) Changes generally include marked consolidation of the cranial and middle lobes with a distinct demarcation between affected and unaffected lungs. Extensive fibrinous pleuritis, pericarditis, and in more chronic cases, abscess formation in the lung, also may be present.

(b) For biotype A septicemia, there is often subcutaneous hemorrhage over the neck and thorax, with edematous lungs and subpleural ecchymosis, but pneumonia is not a feature. There is ulceration and necrosis of the pharynx and esophagus, as well as the occurrence of small necrotic areas on the tips of the abomasal mucosal folds.

(c) The organism can usually be isolated in large numbers from tonsils, lung, liver, and the ulcerated areas of the intestinal tract.

(5) Therapeutic plan. The choice of antibiotic therapy in lambs is based on etiologic probability. P. haemolytica is the most likely pathogen, and the choice of drugs available includes penicillin, ampicillin, tetracyclines, trimethoprim-sulfonamides, and triple sulfas.

(a) Although not all strains of biotype A are sensitive to penicillin, almost all strains are sensitive to **oxytetracycline,** which makes it a suitable first-line drug of choice. An additional advantage is that these drugs are effective against Chlamydia and most *Mycoplasma* species.

(b) Long-acting tetracyclines can be effective, particularly because they can reduce the stresses of handling (only one or two injections are needed); however, tetracyclines are irritating when administered intramuscularly.

(6) Prevention

(a) Vaccination with autogenous and commercial bacterins has not been effective in controlling disease. Specific serotypes or "protective" antigens may be absent from these products.

(b) Prophylactic antibiotics

(i) Feed medication with chlortetracycline or chlortetracycline with **sulfamethazine** has been evaluated as a preventive for pneumonia in range lambs but has met with equivocal results.

(ii) Sulfonamides have been recommended as a preventive for baby lamb pneumonia by intermittent medication of the drinking water of ewes before lambing.

(c) Management strategies

(i) Avoid overcrowding by providing a minimum of 14 square feet per pregnant ewe and a minimum of 4–5 square feet for each lamb.

(ii) The producer should consider **prelambing** shearing, which has an advantage in that housed animals with access to the outdoors are not carrying a high moisture content in their fleeces; thus, the humidity of the barn is lower. Shorn ewes will also seek shelter at lambing, reducing the risk of hypothermia due to exposure in newborn lambs. However, adequate shelter from wind and rain does need to be provided for the shorn ewes.

(iii) During shearing, young sheep should be shorn first, and any cuts should be disinfected and clippers blades sterilized to reduce the risk of spreading *Corynebacterium pseudotuberculosis.* Additionally, avoid dipping or spraying for external parasites if there is any sign of cold wet weather approaching.

b. Other bacterial causes of pneumonia in sheep and goats

(1) ***P. multocida,*** Streptococcus species, ***Escherichia coli,*** and ***Haemophilus*** species may be present in ovine and caprine pneumonia. These bacteria arc

usually secondary invaders. *S.* typhimurium and S. dublin cause diarrhea and abortion in sheep and goats. Respiratory signs may be seen initially.

(2) Francisella ***tularensis*** infection can result in acute illness manifested by high fever, prostration, 'diarrhea, and respiratory signs, including nasal discharge and coughing. Diagnosis is confirmed by the isolation of the causative agent, the presence of Dermacentor andersonii, and a die-off of rodents in the area.

4. Porcine pneumonia. Although individual pigs can be affected with pneumonia on a sporadic basis, there are two forms of infectious respiratory problems that plague the swine industry: **enzootic** pneumonia and pleuropneumonia.

a. **Enzootic** pneumonia

(1) Economic implications. Enzootic pneumonia consistently ranks as the most economically important disease in finishing pigs in North America. Enzootic pneumonia occurs worldwide and has a particularly high incidence in intensive rearing operations. In infected herds, the morbidity rate is highest during the growing period, but the case fatality rate is low. The main adverse effects of this disease include an increase in the need to treat clinical illness caused by secondary bacterial pneumonia and the reduction in feed efficiency and average daily gain for getting the pigs to market.

(2) Clinical findings. The most common form of the disease, as observed in endemically infected herds, begins at 3–10 weeks of age and is insidious in onset.

(a) Initially, the only clinical abnormality is cough in a small proportion of the piglets. This increases such that most animals in the pen show persistent cough, which is particularly obvious at times of activity, such as feeding, and can continue through the entire growing period. The cough is dry and hacking, but signs of respiratory embarrassment are rare, and there is no fever or diminished appetite.

(b) Those pigs that develop signs of pneumonia usually have secondary invasion of the lungs by Pasteurella species or other bacteria. Clinical signs of disease become less obvious with increasing age.

(3) Etiology and pathogenesis

(a) Etiology. The disease is caused by the primary initiator Mycoplasma *hyo*pneumoniae (or *suipneumonia*) with Pasteurella *multocida* as a common secondary invader of the lung. M. hyopneurnonia appears to be host specific, inhabiting the respiratory tract of pigs and surviving in the environment for only a short time. Other pathogens can cause similar pathologic lesions, but this organism appears to be the primary cause of enzootic pneumonia in pigs.

(b) Pathogenesis

(i) Piglets are infected by the mycoplasmal organism early in life, likely from the sow, but also possibly from airborne particles from other pigs.

(ii) M. hyopneumonia causes peribronchiolar lymphoid hyperplasia and mononuclear accumulation in the lamina **propria,** resulting in obliteration of the bronchial lumen. Also, the bronchial mucous glands undergo hypertrophy, and there is hypertrophy of the type II alveolar epithelial cells and progressive loss of cilia on the bronchial mucosa, decreasing the defenses against secondary bacterial infection.

(iii) These damages heal on their own, but if secondary bacterial invasion occurs, more severe pathology occurs, including bronchopneumonia and pleuritis. These complications also cause decrease in feed efficiency and average daily gain.

(4) Diagnostic plan and laboratory tests. The gross and microscopic findings on the lungs of pigs affected with enzootic pneumonia are not pathognomonic; thus, a positive diagnosis requires culture of M. hypopneumoniae from tissues. However, a negative result can often occur because the organism is difficult to culture and is readily overgrown by other nonsignificant organisms, such as

M. hyorhinis. A fluorescent antibody test or an enzyme-linked **immunoperoxi**dase technique may help demonstrate the organism in tissues.

(5) Therapeutic plan. Treatment is usually restricted to individual pigs showing acute respiratory distress, which, being of a secondary bacterial nature, should respond to most broad-spectrum antibiotics.

(a) There is no effective treatment that eliminates infection by M. *hyopneu*monia. However, antibiotics such as **tylosin** tartrate (50 mg/kg) and **tia**mulin (10 mg/kg) orally for 10 days can reduce pulmonary lesions.

(b) Other antibiotics show activity against this mycoplasma, including tetracyclines and the newer fluoroquinolones, such as ciprofloxacin.

(6) Prevention. Because M. hyopneurnonia infects only pigs and transmission requires close pig-to-pig contact, its spread can be limited or even eradicated from a herd.

(a) Eradication is the most satisfactory method of control but requires depopulation of the herd, followed by repopulation with pigs from specific pathogen-free (SPF) herds. Such pigs are commercially available, having been raised in special units populated with the progeny of Caesarianderived piglets.

(b) Separation. **Less** successful methods involve isolating the farrowing area for sows believed free of infection from the rest of the herd, and raising the piglets separately. Alternatively, newborn piglets can be treated with antibiotics effective against Mycopiasma species and removed to isolated premises, with subsequent serologic testing of the breeding herd and culling of seropositive animals. These techniques are far less successful than complete repopulation with disease-free pigs.

b. Contagious pleuropneumonia

(1) Economic implication. This disease is of major economic importance, is worldwide in occurrence, and appears to be increasing in prevalence with the more intensive swine operations.

(2) Patient profile and history. The disease is predominately found in growing pigs, from ages *2* to 6 months.

(3) Clinical **findings**

(a) The disease is characterized by rapid onset and a short course of **severe dyspnea,** the passage of blood-stained foam from the mouth, and a high case-fatality rate.

(b) Clinical course. The disease can be peracute, acute, or chronic, depending on the immune status, and each form is reasonably well defined. The clinical course of the disease in a herd can last several weeks, with new acute cases occurring as chronically affected animals develop a generally unthrifty appearance.

(i) In **peracute** cases, the only sign is sudden death in pigs that may be close to market weight.

(ii) In acute **cases,** severe respiratory distress is observed along with an exaggerated abdominal component (thumps), a reluctance to move, anorexia, and a fever up to **41°C.**

(iii) In more chronic cases, there is fever and anorexia, but the respiratory distress is less severe and a persistent cough develops.

(4) Etiology and pathogenesis

(a) Etiology. The condition is caused by ***Actinobacillus*** pleuropneumonia (formerly known as *Hemophilus* pleuropneumonia), a highly contagious organism that is not isolated from normal porcine respiratory tissues but persists in chronic lesions in the lungs of recovered and apparently healthy pigs, which provide the source for continued infection.

(b) Pathogenesis

(i) It is thought that natural transmission occurs by the aerogenous route, with the source of infection being a subclinically infected or recovered pig. Outbreaks appear to occur in pigs that lack immunity and are overcrowded or subjected to recent stressors, such as large

fluctuations in temperature, recent transportation, or problems with ventilation in the barn.

(ii) When the organism begins to multiply in the lung (within hours of infection), there is rapid development of pulmonary edema and diffuse neutrophilic bronchiolitis and alveolitis. There are also marked vascular effects, which result in infarcts in the lung, thrombosis, and hemorrhage. A hemorrhage, in turn, may result in the pleural inflammation. Pigs appear to die of septic shock.

(5) Diagnostic plan and laboratory tests. The clinical signs in growing pigs of rapid onset and sudden death associated with respiratory signs provides a presumptive diagnosis. Culture at necropsy confirms the infection, and there is a reliable serological test to assess recent infection in the live animals.

(6) Therapeutic plan. Antimicrobial treatment, with drugs such as tetracyclines, spectinomycin, or penicillin, can reduce mortality and improve daily gain in affected pigs. However, the animals treated often continue to remain infected with the organism. Therefore, combined with the peracute losses not possible to prevent, the overall clinical response to treatment can be disappointing.

(7) **Prevention**

(a) Depopulation followed by repopulation with uninfected pigs is the only effective control method for this infection. The all-in, all-out system of feeding and marketing pigs can help by reducing the introduction of new stock to the herd.

(b) Management practices that can reduce the impact of this disease should emphasize the raising of weaned pigs in pens that are separate from the older stock in the herd.

(c) Vaccination is effective in reducing mortality, but vaccinated animals can still be carriers. A major problem plaguing the production of an effective vaccine is the large number of serotypes of *Actinobacillus pleuropneumonia*, against which effective serotype-specific vaccines incorporating all the important antigens have yet to be produced.

C. Parasitic pneumonia

1. Equine parasitic pneumonia

a. Lung worms

(1) Patient profile and history. The donkey is the natural host of the equine lungworm *Dictyocaulus* amfieldi. Horses become infected when they graze pastures with infected donkeys or previously contaminated pastures. The common complaints include a persistent cough, increased respiratory rate, and forced expiration in horses. Donkeys usually show no signs even with heavy infestation.

(2) Clinical findings. The predominate clinical sign is a chronic cough. Horses are afebrile, and their appetites are unaffected.

(3) Etiology and pathogenesis. The larvae migrate through the gut wall and are carried hematogenously to the lungs.

(a) In the donkey, the larvae mature in the bronchi and lay eggs. The eggs are coughed up and swallowed.

(b) In the horse, the maturation of the larvae in the airways is retarded, and the worms remain immature; thus, the infection does not become patent. Lung pathology is limited to the caudal lobes. There is epithelial hyperplasia with an increase in size and number of goblet cells. Grossly, there are areas of overinflation, mucous exudate, and coiled worms.

(4) Diagnostic plan and laboratory tests. Clinical signs of lungworm infestation must be differentiated from chronic obstructive pulmonary disease (COPD).

(a) Clinical suspicion. Exposure to donkeys on pasture is suggestive of lungworm infestation, and although this infestation is not patent in horses and is fecal Baerman-negative, it may be useful to perform fecal sedimentation on any closely housed donkeys.

(b) A transtracheal aspirate shows neutrophils and eosinophils, and bronchoalveolar lavage may recover intact lungworms.

(5) Therapeutic plan. Anthelmintics, such as fenbendazole (30 mg/kg) or ivermectin (200 μg/kg orally), are effective treatment. Animals may initially worsen with treatment because of the death of larvae causing an intense inflammatory response. Therefore, affected horses may benefit from concurrent treatment with anti-inflammatory agents, such as nonsteroidal anti-inflammatory drugs (NSAIDs), or a glucocorticoid.

(6) Prevention. Donkeys should be treated with appropriate anthelmintics, and pastures should be rotated. In temperate regions, the parasites are unableto survive during winter on pasture. Most routine broad-spectrum anthelmintic regimens for intestinal parasites also control lungworms in hones.

b. Parascarid migration in foals

(1) Patient profile and history. This disease occurs in foals and weanlings, with cough as the main complaint.

(2) Clinical findings. Signs consist of a transient episode of coughing accompanied by a mucopurulent nasal discharge. Throughout the episode, the rectal temperature remains normal, and there is seldom sufficient damage in the lungs to cause a noticeable increase in the respiratory rate or depth.

(3) Etiology and pathogenesis. The disease is caused by the ascarid *Parascaris equorum* in the course of its development as an intestinal parasite of foals. When foals are infected with *P. equorum* eggs through ingestion, the larvae penetrate the gut wall and undergo a hepatic–tracheal migration. The larvae arrive by a hematogenous route and then migrate up the airways and return to the intestine to mature. Their presence in the airways stimulates mucus production. Signs are usually transient.

(4) Diagnostic plan and laboratory tests. As in lungworm infections, a transtracheal aspirate or bronchoalveolar lavage may show eosinophils present in the cytology. However, as the clinical signs are usually mild and the clinical course transient, such diagnostic procedures are seldom performed.

(5) Differential diagnoses. This infection must be differentiated from other causes of coughing in foals, particularly the far more prevalent viral respiratory infections.

(6) Therapeutic plan. Most of the commonly used anthelmintics for horses are effective against adult *P. equorum,* with ivermectin also being effective against the larval stages.

2. Bovine parasitic pneumonia

a. Patient profile and history. This disease occurs most commonly in dairy calves that are younger than 1 year of age in the summer and fall of the first season at pasture. Clinical disease may also occur in adult cattle that have minimal prior exposure to the parasite and have recently moved onto heavily contaminated pasture.

b. Clinical findings

(1) Acute form. In acute cases, severe verminous pneumonia occurs. Acute cases progress quickly, and death from progressive respiratory failure can occur in 3–14 days. This form of the disease occurs in calves 1–2 weeks after being moved to heavily contaminated pasture, and many calves are affected simultaneously.

(a) There is a sudden onset of rapid shallow breathing with a marked abdominal component. Accompanying this is a frequent deep cough and a fever that may reach 41°C.

(b) On auscultation, all portions of the lung are affected with increased bronchial tones and fine crackles. The animals can remain reasonably bright and active and attempt to eat, although severe respiratory distress may prevent eating.

(2) Subacute form. The more common form of the disease is a subacute verminous pneumonia, which has a prolonged clinical course of 3–4 weeks.

(a) In these cases, the onset is also sudden, with an increased respiratory rate

(60–70 beats/minute), frequent paroxysms of coughing, and, in severe cases, an expiratory grunt. The body temperature, however, is normal or only slightly elevated. There may also be evidence of recent **diarrhea**.

(b) On **auscultation** of the lungs, there are crackles and wheezes bilaterally and some areas of ventral dullness on chest percussion, suggesting pulmonary consolidation.

(c) Affected animals **lose weight rapidly**, and although the mortality rate is much lower than in the acute form, surviving calves have severely damaged lungs that result in labored breathing for several months. In addition, these calves are predisposed to secondary bacterial pneumonia or a proliferative pneumonia of possible allergic origin.

c. **Etiology and pathogenesis**

(1) **Etiology.** Both forms of the disease are consistent with bronchopneumonia caused by the pulmonary reaction to the invasion of the larvae of ***Dictyocaulus viviparus***.

(a) The **acute form** is likely the result of massive invasion of larvae.

(b) Moderate infestations lead to the **subacute form**, with light infestations resulting in few clinical signs.

(2) **Pathogenesis**

(a) **Transmission.** Pastures are contaminated with the infective third stage larvae. These larvae develop in feces at pasture, and although they are inactive, the larvae are spread by

(i) Diarrhea

(ii) Rain

(iii) A high concentration of animals

(iv) Earthworms

(v) The propelling of larvae in the explosive discharge (up to 3 m spread) of the fungus *Pilobolus*

(b) **Route of infection**

(i) The infective larvae are ingested by the susceptible animal and migrate from the intestinal tract, via the lymphatics and venous circulation, to the alveoli (1–7 days).

(ii) After a prepatent phase of 7–25 days, larvae mature in the bronchi and trachea and release eggs, which are coughed up, swallowed, and passed in the feces as larvae (having hatched in the intestine). Larvae are resistant to freezing and can survive the winter on pasture, particularly in cool, moist areas where herbage is long.

(iii) The adult worms survive in the bronchi for approximately 7 weeks, by which time immunity develops, and there is self-cure because most worms die or are discharged.

(c) The **pathology** found is that of **lung lobe consolidation** and an eosinophilic and macrophage response to aspirated eggs and new larvae. Parasitic infection and *the* subsequent inflammation predisposes the lung to secondary bacterial infection. Therefore, parasitic and secondary bacterial pneumonia may subsequently be difficult to differentiate.

(d) There is also a **reinfection syndrome** in which adults with immunity are exposed to massive numbers of infective larvae. These cattle can develop respiratory disease solely because of their immune reaction, with lymphoid proliferation around dead larvae, no adult worms at necropsy, and no eggs released during the course of the illness.

d. **Diagnostic plan and laboratory tests**

(1) **Clinical suspicion.** When clinically apparent, parasitic pneumonia may be difficult to differentiate from bacterial or viral pneumonia. However:

(a) Failure to respond to standard treatment for these conditions and disease occurring at pasture in the summer or fall support a diagnosis of verminous pneumonia.

(b) A **useful clinical feature** of verminous pneumonia is that the associated cough is relatively soft and paroxysmal rather than the harsh, dry cough of viral pneumonia.

(2) Necropsy. In areas of outbreaks, deaths are also frequent enough to allow postmortem diagnosis, with the adult worms readily observed in the airways by the naked eye.

(3) Laboratory studies

(a) A transtracheal aspirate or bronchoalveolar lavage shows greatly increased numbers of eosinophils and possibly ova or larvae in the sample.

(b) Fecal sedimentation of affected animals should be examined by the **Baerman technique** for the presence of **lungworm** larvae.

e. Therapeutic plan

(1) Most modern, broad-spectrum anthelmintics [e.g., albendazole at 7.5 mg/kg orally, levamisole (13.6%) at 8 mg/kg subcutaneously, fenbendazole at 5 mg/kg orally] are active against all stages of *D. viviparus*. **Ivermectin** (0.2 mg/kg subcutaneously) is particularly effective against the immature and mature stages, even at one-fourth of the recommended dose. With the full recommended dose, residual protection is provided for up to 28 days.

(2) The topical formulations of both levamisole and ivermectin are also effective. However, killing the parasite does not resolve the damage already present in the lungs.

f. Prevention

(1) Management strategies. Much of the prevention rests on pasture management, such as preventing overcrowding and avoiding continuous use of the same pasture for young stock. Wet, swampy pastures allow maximal larval development and, therefore, should be used for grazing only the adult (immune) animals.

(2) Deworming. After winter housing, yearlings should be dewormed before release onto pasture so that they do not begin a new cycle of pasture contamination.

(3) Vaccination. In Europe, a vaccine is available that consists of irradiated larvae that cannot mature. This vaccine is administered orally to calves before they are turned out to pasture. This primes the immune response in advance of any exposure to natural infection.

3. Ovine and caprine parasitic pneumonia

a. Etiology. Lungworms that affect sheep and goats include ***Dictyocaulus filaria, Protostrongylus rufescens,*** and ***Müellerius capillaris.*** Each organism is capable of causing varying degrees of verminous pneumonia, but the former two appear to be of greater clinical significance.

(1) *D. **filaria*** is usually found in the posterodorsal region of the diaphragmatic lobes and may lead to secondary pneumonia and pleuritis. Although it is primarily a parasite of sheep, it is highly pathogenic to young goats.

(2) *P. **rufescens*** invades small bronchioles and may lead to secondary pneumonia and pleuritis.

(3) ***M. capillaris*** infections in adult sheep usually result in nodular or diffuse lesions in the subpleural parenchyma and have been considered to be of minimal significance. However, recent reports suggest that this parasite may cause widespread interstitial pneumonia in goats, with affected animals gradually losing condition.

b. Clinical **findings**

(1) *D. **filaria*** infection produces a cough that results from bronchial irritation, along with moderate dyspnea and loss of condition. Extreme dyspnea occurs if most airways become plugged with debris.

(2) *P. rufescens* infestations cause clinical signs similar to those of *D. filaria,* with only the kids and lambs showing serious clinical involvement. In contrast to the direct life cycle of *D. filaria, P. rufescens* is indirect, requiring a land snail for the second stage of larval development. Because of this, massive infestations are unlikely to occur.

(3) Infection with *M. **capillaris*** is relatively innocuous clinically but may constitute a limiting factor in the production of choice lambs.

c. Diagnostic plan and laboratory tests. The main method of diagnosis is Baerman examination of the feces for the larvae of each lungworm parasite.
d. Therapeutic plan
(1) Most of the broad-spectrum anthelmintics such as albendazole (3.8 mg/kg), fenbendazole (5 mg/kg), ivermectin (0.2 mg/kg), or levamisole (8 mg/kg), are effective against *D. filaria.* Few of these drugs have been tested against *P. rufescens,* but they are likely effective.
(2) M. *capillaris* is more difficult to treat, with anthelmintics usually only effective against the adult forms. Efficacy against all forms of the parasite requires treatment with products such as fenbendazole or albendazole in the feed for 2 weeks.
e. Prevention. Preventing parasitic pneumonia by pasture rotation is difficult. Larvae of *D. filaria* can withstand long periods of freezing. Snails, which are the intermediate hosts of *P. rufescens* and *M. capillaris,* are particularly prevalent in poorly drained pastures. Fencing of wet areas of pastures may be beneficial.

4. Parasitic pneumonia in swine
a. Patient profile and etiology. Parasitic pneumonia occurs in pigs that are raised in management systems that allow access to earthworms and is most prevalent in pigs ages 4–6 months. Lungworms that infest pigs include *Metastrongylus* apri (most common), *M. salmi,* or *M.* pudendotectus, with mixed infections possible.
b. Clinical findings. Clinical cases show a barking cough that is easily induced by exercise, resulting from the parasitic bronchitis. In severe cases, pneumonia, poor growth, and debilitation can occur, but minimal clinical signs are apparent after experimental disease.
c. Pathogenesis. The parasite lays eggs in the lungs of the pig. The eggs are then coughed up, swallowed, and passed in the manure. The embryonated eggs or larvae are eaten by earthworms and develop successively to second- and third-stage larvae. Reinfection occurs when the earthworm is eaten by other pigs.
d. Diagnostic plan. Necropsy is usually the method of diagnosis.
e. Therapeutic plan. Many of the broad-spectrum anthelmintics are effective, including levamisole (8 mg/kg) in the feed, mebendazole for 2 days successively in the feed (15 mg/kg), or a single injection of ivermectin.
f. Prevention
(1) Pigs that run in dirt yards or at pasture should be moved at short intervals to prevent the ingestion of infected earthworms.
(2) Rooting by pigs can be prevented by providing adequate feed and by applying nose rings.
(3) Pastures that are known to be contaminated should not be restocked for at least 6 months.

D. Aspiration pneumonia

1. Patient profile and history. Aspiration pneumonia can occur in any species, and the outcome is largely dependent on the nature of the material aspirated. In horses and cows, this disease can occur as a result of misdirection of a stomach tube and subsequent introduction of fluid destined for the intestinal tract. Horses are also at risk for aspiration pneumonia in cases of choke (esophageal obstruction) or secondary to pharyngeal paralysis associated with guttural pouch mycosis. A severe form of aspiration pneumonia can occur in cows with third-stage milk fever, where regurgitation and aspiration of rumen contents can occur.

2. Clinical findings
a. The most severe form of aspiration, as occurs in cattle with rumen content aspiration, is a necrotizing pneumonia that progresses to pleuritis. There is also toxemia with cardiovascular collapse and rapid death. In these cases, breath odors are usually extremely foul, indicative of the necrotic lung tissue.
b. When much smaller quantities of material are aspirated (e.g., following choke or pharyngeal paralysis in a horse), the signs of pneumonia are less severe, with varying degrees of respiratory distress, fever, depression, and cough.

3. Diagnostic plan and laboratory tests
a. A diagnosis is often apparent from the history, such as signs of pneumonia closely following recent unsuccessful attempts at oral or gastric medication. Additionally, food material at the external nostrils can indicate pharyngeal dysfunction.
b. If the animal is not in severe respiratory distress, endoscopic examination of the trachea can confirm the diagnosis by observing food material in the lower trachea.
c. Other diagnostic aids include chest radiographs, which vary greatly in appearance depending on the many circumstances associated with the aspiration. Transtracheal aspirate cytology may show plant material, and the culture results are often a mixture of gram-positive, gram-negative, and anaerobic bacteria.

4. Therapeutic plan
a. When aspiration has occurred, treatment is used to control infection and inflammation as the lung defenses deal with neutralizing and expelling the foreign material.
(1) Broad-spectrum antibiotics that are effective against anaerobic bacteria are recommended (e.g., combinations of penicillin with an aminoglycoside, or potentiated sulfonamide).
(2) NSAIDs, such as flunixin meglumine, can help reduce lung inflammation, and cardiovascular support with intravenous fluids is indicated where there are signs of toxemia. Pleuritis is often present in severe cases and may require drainage of the pleural fluid (see I E).
b. Other treatment measures are dependent on the associated cause, such as choke, milk fever, or pharyngeal dysfunction, which should be corrected concurrently if possible.

5. Prognosis. The prognosis for recovery from severe aspiration pneumonia is grave. Although less fulminant cases of aspiration pneumonia are often readily responsive to treatment, a successful outcome is more dependent on resolving the underlying disorder (e.g., choke, pharyngeal paralysis or dysfunction).

E. Pleuritis and pleural effusion

1. Introduction
a. Pleural surfaces. The pleural surfaces of the thorax are composed of the visceral and parietal pleura.
(1) The visceral pleura, which covers the lung surface, lacks specific pain receptors.
(2) The parietal pleura, which lines the chest wall, diaphragm, and mediastinum, contains pain receptors; thus, when the pleural lining is inflamed, it can be a source of significant pain for the animal.
b. Mediastinum
(1) In cattle, sheep, goats, and pigs, the mediastinum, which separates the right from left pleural spaces, is intact. In species with a complete mediastinum, disease processes (e.g., pneumothorax, pleuritis) may be restricted to one pleural space, with the opposite lung and pleural space unaffected.
(2) The bone has a thin mediastinum that is frequently perforated. The same disease processes in horses can move from affected to unaffected sides of the thorax.
c. Pleural fluid. The visceral and parietal pleura are in close contact with each other. This proximity creates a potential space that is lubricated by a small amount of pleural fluid in which equal amounts are produced and then resorbed. Effusion in large quantities arises mainly when there is increased vascular permeability and reduction of lymphatic drainage, as occurs in inflammation of the pleura.

2. Patient profile and history. The history is variable.
a. In horses, this disease often is associated with stressful events, such as recent transport over long distances or a stressful competition following viral respiratory disease.
b. In other species such as cattle, pleuritis can be part of the pneumonia complex of *Pasteurella* infection (see I B 2).

3. Clinical findings
   a. Pain
      (1) Animals with acute pleuritis show chest pain and are sensitive to touch over the thorax.
      (2) Other signs of pain include abduction of the elbows, reluctance to lie down, and a splinted abdomen. In horses, these signs are sometimes misinterpreted as a sign of colic.
   b. Accompanying signs can include the presence of guarded, shallow respiration with a shallow cough and a nasal discharge, which may have a fetid odor. Anorexia, depression, and fever are usually present. Ventral edema in the brisket area, when present, is a hallmark of the effects of the pleural inflammatory process.
   c. In chronic cases, the signs may be less obvious, with weight loss, anorexia, depression, or ventral edema being the only significant clinical findings.
      (1) On auscultation of the lungs, there is usually an absence of lung sounds ventrally, which is accompanied by widely radiating heart sounds.
      (2) Pleural friction rubs may be detected but are surprisingly not a consistent finding in pleuritis.
      (3) There is ventral dullness on percussion of the chest.
   d. In acute cases in the horse, ventral dullness is often associated with a horizontal fluid line. Because the disease in cattle is most often a component of diffuse fibrinous pleuropneumonia, no discrete horizontal line is to be expected on chest percussion.

4. Etiology
   a. **Equine** pleuritis results from the stress of transport, trauma of perforating thoracic wounds, esophageal perforation, or, less commonly, lymphosarcoma or accompanying equine infectious anemia. Solely infectious causes also are sporadically reported and include infections by Mycoplasma *felis* or Nocardia species.
   b. Bovine pleuritis is usually secondary to *Pasteurella* pneumonia or traumatic reticuloperitonitis. An infectious cause of bovine pleuritis, contagious bovine pleuropneumonia (Mycoplasma mycoides var mycoides), caused large losses in the North American cattle population when the continent was being settled, but this has been eradicated from the continent.
   c. Porcine **pleuritis.** Pigs have pleuritis and pleural effusion as part of actinobacillosis and in polyserositis of Glasser's disease caused by Hemophilus suis or *parasuis* infection, which also results in acute arthritis, peritonitis, and pericarditis (see Chapters 8 and 13).

5. Diagnostic plan and laboratory tests
   a. The clinical signs of systemic illness, accompanied by chest pain and shallow breathing, are highly suggestive of pleuritis. Thoracic percussion in the acute phase is suggestive of fluid within the chest.
   b. Of the laboratory tests available, both hematology and routine biochemistry are nonspecific. A neutrophilia with or without left shift and possibly an anemia of chronic infection may be noted on hematology, and low albumin accompanied by elevations in fibrinogen and globulins are observed on biochemistry.
   c. More definitive tests for diagnosis include radiology, ultrasonography, and thoracocentesis.
      (1) On chest radiographs, there is a pleural effusion line and often signs of an accompanying pneumonia, pulmonary abscess, or both. With adult horses and cows, this finding is often restricted in value because the large chest size reduces the diagnostic quality of the radiographs.
      (2) Chest ultrasound is highly sensitive for the detection of pleural fluid and can provide information regarding pleural thickening, loculation of pleural fluid, and the presence of fibrin. Also, hyperechoic echoes in the pleural fluid can suggest the presence of gas bubbles caused by anaerobic bacteria. These findings dictate highly specific therapy and an unfavorable prognosis.
      (3) Thoracocentesis is often key in assisting in diagnosis, therapy, and prognosis.

      (a) Fluid recovered varies from a clear yellow transudate in milder cases to cloudy and even purulent fluid in more severe cases.
         (i) Normal pleural fluid is difficult to obtain by thoracocentesis and has a cell count of less than 10,000/μl white blood cell count (10×10^9/L). Although cell counts can increase greatly in pleuritis, there is a large range in cell counts, and there has been no correlation of pleural fluid cell numbers with survival.
         (ii) Fluid with a foul odor suggests an anaerobic infection, such as necrotizing pneumonia.
      (b) The pleural fluid should be cultured for bacteria and should have a Gram stain performed to give an initial guide to treatment. Frequently, there is no growth from this fluid, and a culture and sensitivity from a transtracheal aspirate is then indicated, as many cases of pleuritis have an underlying pulmonary problem or possibly began as pneumonia.

6. Therapeutic plan. Chest drainage, appropriate antibiotics, and supportive nursing care are required for horses with pleuritis. The other large animal species seldom receive similar Intensive care; thus, this discussion focuses on the horse. Should treatment be required, similar methods can be used for specific treatment of pleuritis in other species.
   a. Drainage of pleural fluid is important if the process in the chest is highly purulent.
      (1) Continuous drainage can be managed by using indwelling chest drains with a one-way Heimlich valve, but this is a labor-intensrve venture and may not be practical, particularly in many stable environments. This method is associated with complications, such as cellulitis and possible pneumothorax.
      (2) Alternatively, repeated thoracocentesis can be performed every several days until the pleural inflammation subsides with treatment. In chronic pleuritis, fibrous adhesions often impair complete drainage.
   b. Antibiotics. Selection of the appropriate antibiotic for treatment is initially based on Gram-stain results.
      (1) In horses, sodium penicillin (20,000 IU/kg intravenously every 6 hours) and gentamicin (2.2 mg/kg intravenously every 8 hours) are commonly chosen because this combination is effective against most gram-positive, gram-negative, and non-Bacillus *fragilis* anaerobic bacteria.
      (2) In circumstances in which B. *fragilis* is involved, (penicillinase producers) metronidazole should be added to the regimen (25 mg/kg orally every 12 hours).
      (3) Alternatively in horses, chloramphenicol (25 mg/kg orally every 6 hours) can be used as a sole treatment. Chloramphenicol should not be used in food-producing animals.
   c. Supportive care. Stress should be minimized. Rest and fluids should be provided as required. Pain relief using NSAIDs (e.g., phenylbutazone, flunixin meglumine) may help the animal regain its appetite and increase its comfort.
   d. Additional treatment
      (1) Heparin (40 IU/kg subcutaneously every 12 hours) may decrease adhesion formation.
      (2) For chronic, one-sided pleuropneumonia, rib resection and thoracotomy to drain purulent material has been a successful salvage procedure.

7. Prognosis. On initial assessment, the prognosis is often poor, particularly for return to performance. Also, pleuritis is very expensive to treat. However, there is a wide range in recovery, from respiratory cripples to apparently complete resolution. The prognosis is guarded in anaerobic and gram-negative infections with accompanying pneumonia, pulmonary abscess, or both. Conversely, some horses with mild pleural effusion appear to have full recovery. Unfortunately, no one laboratory parameter accurately determines the prognosis.

F. Noninfectious respiratory diseases

1. Metastatic pneumonia (also called vena cava syndrome)
   a. Patient profile and history. This disease occurs in cattle older than 1 year and can

occur in any breed, sex, or class of cattle. The main complaints may include weight loss, respiratory disturbance, or occasionally thoracic pain.

b. **Clinical** findings

(1) Affected cattle with a classic presentation of this syndrome have tachycardia, tachypnea, and expiratory dyspnea with groaning and wheezes over much of the chest. Accompanying these signs are epistaxis, hemoptysis, pale mucous membranes, and hemic murmurs. The combination of anemia, widespread wheezes, and hemoptysis is generally regarded as pathognomonic for this disease. The affected animal often has a history of weight loss and cough for weeks to months, but in some cases, the signs may be acute.

(2) Other clinical signs include fever, thoracic pain on deep palpation of the sternum, hepatomegaly, subcutaneous emphysema, froth at the muzzle, and melena caused by swallowing the blood that is being coughed up.

c. Etiology and pathogenesis

(1) This disease is thought to develop from an initial **rumenitis** secondary to lactic acidosis (see Chapter 3 1 A 2). As a result of the chemical damage to the rumen epithelium, bacteria (*e.g., Fusobacterium necrophorum, Actinobacillus* pyogenes) are able to penetrate the rumen epithelium to be transported to the liver portal drainage system, where they are filtered and cause liver abscesses. If an abscess is located adjacent to the caudal vena cava, it may result in the development of septic emboli within the caval vein. This condition then showers the lungs and causes pulmonary arterial thrombosis and pulmonary abscessation, along with pulmonary hypertension and aneurysm formation.

(2) The eventual hemoptysis that is so clinically distinctive is the result of erosion of a pulmonary abscess into an arterial wall, rupture of a pulmonary aneurysm, or both. Other signs, such as anemia, hemic murmur, melena, and widespread wheezes are directly related to the massive lung bleeding.

d. **Diagnostic** plan and laboratory tests. In patients with the pathognomonic signs, usually no further diagnostic tests are required.

(1) The CBC shows anemia and a neutrophilic leukocytosis. ***Serum*** chemistry may have hyperglobulinemia and liver enzyme changes (elevated aspartate aminotransferase, γ-glutamyl transferase) merely reflective of passive congestion.

(2) Chest radiography usually shows only an irregular increase in lung density, but in some cases, there may be more definitive changes, including small discrete densities indicative of embolic infarction and collapse or large spherical densities with cavitating nodules and gas or fluid interfaces.

e. Therapeutic plan

(1) The case fatality rate is usually 100%. Thus, when the diagnosis is established, treatment is rarely indicated.

(2) In valuable animals, supportive treatment can be undertaken, such as blood transfusion in the acute stage, along with **furosemide** (0.4–1 .1 mg/kg intravenously or intramuscularly twice daily) and **flunixin meglumine** (0.5–1.1 mg/kg intravenously or intramuscularly given one to three times daily) as needed for the dyspnea. For the organisms usually involved, penicillin is the drug of choice, with a dose of 22,000 U/kg intramuscularly twice daily for extended periods (weeks to months).

f. Prevention. Because the initiating basis of this disease is rumenitis with subsequent liver abscess formation, measures to reduce the possibility of developing vena cava syndrome include:

(1) Slowing the introduction of high-energy rations to the cattle

(2) Feeding antibiotics during the periods of increase in concentrate feed

2. Chronic obstructive pulmonary disease (COPD)

a. Patient profile and history

(1) This is a worldwide disease of horses that are usually more than 5 years of age and is seen more frequently in stabled animals.

(2) Clinical signs associated with COPD (also known as chronic airway disease, heaves, broken wind, emphysema, chronic bronchiolitis, or recurrent airway obstruction) are usually exacerbated by poor environmental conditions, such as poor ventilation, overcrowding, dusty stables, or breathing molds from stored feeds.

b. Clinical findings

(1) The main complaint is a chronic cough and sometimes an associated exercise intolerance. The cough is usually worse when the hone is stabled. Other common signs include bilateral mucopurulent nasal discharge, dyspnea characterized by nostril flaring, an abdominal lift to the expiration, abdominal muscle hypertrophy (heave line), and pumping of the anus on respiration.

(2) On examination of the lung fields, there are often crackles, wheezes, or both on auscultation, and expanded caudal lung borders with hyperresonance on chest percussion.

(3) Affected horses are afebrile and usually maintain an excellent appetite and demeanor, unless the increased effort of breathing is severe enough to interfere with eating. In such circumstances, weight loss may also occur.

c. Etiology and pathogenesis

(1) Etiology. Proposed instigating factors for horses developing **COPD** include previous respiratory viral infection, allergies to dust and fungal spores, dietary factors such as ingestion of 3-methylindole, and, in some horses, genetic predisposition.

(2) Pathogenesis

(a) Irrespective of the initiating cause, the affected horse develops excessive pulmonary reactivity to illdefined airborne allergens, which, when present in sufficient concentration in the ambient air, induce clinical signs. Conversely, affected horses can be relatively free of any clinical signs when not exposed to these allergens and in dust-free settings.

(b) The structural changes seen in the lungs of affected horses with these clinical signs vary from chronic bronchiolitis with diffuse epithelial hyperplasia and mucus plugs to acinar overinflation and peribronchiolar fibrosis and cellular infiltration.

d. Diagnostic plan and laboratory tests

(1) Clinical signs of chronic recurrent cough in an older horse associated with stabling and no systemic sign of illness is usually sufficient for establishing a diagnosis.

(2) Laboratory studies. The CBC is almost invariably normal. Transtracheal aspirate or bronchoalveolar lavage cytology reveals large numbers of non-degenerative neutrophils, which are present in the lower airways because of immune stimulation rather than bacterial infection.

(3) Atropine challenge. Hones can be tested pharmacologically for the presence of airway spasm by administering atropine (0.022 mg/kg intravenously). Resolution of clinical signs of dyspnea and improvement of lung sounds constitutes a positive response. These results also determine the proportion of the clinical signs caused by reversible bronchospasm, which can be treated with bronchodilator treatment.

(4) Arterial blood gas **determination** and pulmonary function testing. A resting arterial oxygen tension (PaO_2) of less than 83 mm Hg and a maximum change in intrapleural pressure with a tidal breath of greater than 6 mm Hg are suggestive.

(5) Chest radiographs may demonstrate prominent bronchial and interstitial markings but seldom contribute substantially to diagnosis or disease management

e. Differential diagnoses. The differential diagnoses can include bacterial pneumonia, pulmonary neoplasia, and diffuse restrictive diseases. Radiography and lung biopsy are the primary tools for these differentiations.

f. Therapeutic plan

(1) Management strategies

(a) Improvement of air quality in the horse's environment is the most important factor in managing the clinical signs and decreasing the progression of the disease. The horse should spend as much time as possible outside on pasture.

(b) During any necessary period of stabling, the stall should have ample fresh air (e.g., next to a door or window), and the bedding should be as dust free as possible (e.g., peat moss, shredded newspaper). Hay should be thoroughly soaked in water or the feed changed to a complete pelleted ration. Special moist silage for horses is also commercially available and quite effective in minimizing dust from the feed source. Environmental changes must be complete and permanent, otherwise clinical signs will rapidly recur following lapses in dust control.

(2) Medications include bronchodilators for the bronchospasm, expectorants to aid in decreasing the mucus buildup in the airways, and corticosteroids to decrease airway inflammation.

(a) Commonly available bronchodilators include clenbuterol (available in Canada only), aminophylline-theophylline, ephedrine, and antihistamines.

(i) Aerosol therapy is now possible with products such as terbutaline (a β_2 agonist) or ipatromium bromide (parasympatholytic or atropine like in action), in conjunction with recent production of medicating face masks for horses.

(ii) Though not a bronchodilator, the mast-cell stabilizer cromolyn sodium also can be administered by the aerosol route and is effective if given to horses that have a component of immediate (type I) hypersensitivity to their airway reactivity.

(b) **Expectorants,** which include iodides, glyceryl guaiacolate, or simple nebulization with saline, are often used as adjunctive treatment but are of lesser benefit alone in resolving clinical signs.

(c) Corticosteroids are reserved usually for horses that are not responsive to the previous treatment. Although their full effects on the lungs remain poorly understood, corticosteroids do reduce airway inflammation and promote airway smooth-muscle relaxation. Prednisone can be given for a severe episode at 1 mg/kg orally twice daily with tapering doses by half every sixth or seventh day and discontinued entirely when the environmental challenge is solved. Some horse may benefit from prolonged administration of low levels of corticosteroids (prednisone 0.5 mg/kg orally every 48 hours).

(d) Antibacterials also may be indicated in selected horses that also have signs of airway sepsis on transtracheal aspiration or systemic signs of bacterial pneumonia. In most North American horses with COPD, antibiotics are seldom indicated or necessary.

3. Hypersensitivity **pneumonitis** (extrinsic **alveolitis,** bovine farmers' lung)

a. Patient profile and history. This disease occurs almost exclusively in housed adult cattle (particularly dairy cattle) that are fed stored roughage feed. Although the disease occurs sporadically, it appears mainly in the fall, winter, and early spring, when cattle are confined.

b. Clinical findings

(1) The main complaint is usually the increasingly progressive respiratory distress and coughing, along with anorexia and decreased milk production in an individual cow.

(2) On clinical examination, the affected animal shows tachypnea, expiratory dyspnea and a dry, nonproductive cough. There may be a thick nasal discharge, and on chest auscultation, there are increased bronchial sounds and craniioventral crackles. Fever may also be present but it is usually transient.

c. Etiology and pathogenesis. The respiratory distress is attributable to a hypersensitivity reaction to thermophylic molds (*Micropolyspora* faeni and Thermoactinomyces vulgaris) that contaminate roughage feeds. Antigen exposure in sensitized individuals activates cellular and humoral immune responses. The pulmonary inflammation results from complement activation, histamine release, and polymorphonuclear neutrophil and macrophage recruitment. The resulting pathology is interstitial

infiltration by lymphocytes, plasma cells, macrophages, and granuloma formation. Healing results in restrictive fibrosis.

d. Diagnostic plan and laboratory tests

(1) On transtracheal aspirate, there are neutrophils and macrophages in the cytology but no bacteria unless there is a secondary bacterial pneumonia. It is the absence of these signs of infection that suggests consideration of this immune mediated disease.

(2) Suspect cattle can have serum samples analyzed for the presence of precipitins to the antigens of M. faeni and *T. vulgaris*. Also, the *M.* faeni antigen can be administered intradermally, and a positive reaction may be noted as a local Arthus reaction after 4–6 hours.

e. Therapeutic plan. Antihistamines and corticosteroids may provide some relief for individual animals, but when the respiratory signs are noticed, the pulmonary damage is usually irreversible.

f. Prevention. When the producer recognizes the problem, the only change possible in management (if the cattle must be housed for extended periods) is to minimize antigen exposure by feeding hay outside or by feeding silage.

4. Acute respiratory distress syndrome [ARDS, acute **bovine** pulmonary emphysema and edema **(ABPE),** fog **fever]**

a. Patient profile and history. This severe type of respiratory distress occurs in late summer and fall mainly in adult beef cattle (ages 3–8 years) that have a history of having been moved from poor to lush pasture in the previous 1–2 weeks. Geographically, this disease appears to be most prevalent in the western part of North America.

b. Clinical findings

(1) The main complaint is usually an acute onset of severe dyspnea or open-mouth breathing, expiratory grunt, and tachypnea in mature cattle, often with several to many in a herd affected simultaneously. Although the heart rates are usually elevated, fever (suggestive of an infectious cause) is not usually a prominent feature.

(2) The mucous membranes can become cyanotic as respiratory embarrassment advances, and **subcutaneous** emphysema over the shoulders and thoracic inlet may appear as a consequence of the severe respiratory effort. There may even be some cattle found dead with few premonitory signs.

(3) In acute cases, there are usually increased bronchial tones over the ventral aspects of the lungs, but the dorsal portions of the lungs are surprisingly silent, with a relative absence of breath sounds despite the obvious respiratory distress. Nursing calves running with the cows are usually completely unaffected.

c. Etiology and pathogenesis

(1) This disease is an interstitial pneumonia with lung changes of pulmonary edema and interstitial emphysema, which is the result of ruminal transformation of dietary L-tryptophan to more toxic 3-methylindole (3-MI).

(a) A **sudden** feed change to lush pasture or brassica plants (e.g., rape, kale, tops of turnips) results in the overgrowth of ruminal lactobacillus, which produces toxic levels of 3-MI from dietary L-tryptophan. The ruminally produced 3-MI is then absorbed into the circulation and is metabolized by microsomal enzymes in the Clara cells of the lung to pneumotoxic metabolites that injure the alveolar and capillary epithelium.

(b) Consequently, there is plasma transudation in the exudative stage. If the animal survives, there is a proliferation of type II pneumocytes, alveolar epithelialization, and irreversible fibrosis. At necropsy, there is subpleural and interstitial emphysema, alveolar edema, epithelial hyperplasia, and hyaline membrane formation.

(2) There are several other causes of ARDS, with the clinical and pathologic pictures being indistinguishable from the mechanism described in I F 4 c (1). These other causes include:

(a) Pulmonary damage by mixed function oxidase metabolism of other

xenobiotics (e.g., 4-ipomeanol, a toxin from sweet potatoes infested with the mold *Fusarium solani*)

(b) ***Perilla*** ketona, a pneumotoxic principle found in leaves and seeds of purple mint *(Perilla frutescens);* there may be a mint-like odor to the edema fluid as noted at necropsy

(c) Inhalation of toxic gases, such as the manure gases (H_2S, ammonia, methane) or nitrogen dioxide from silos

d. Diagnostic plan. Diagnosis usually rests on the clinical history and risk factors for the possible causes, coupled with the signs of an acute onset of severe respiratory distress without fever ortoxemia.

e. Differential diagnoses. Additional causes of acute respiratory distress in cattle that appear to be similar in clinical appearance to ARDS include acute immune reactions such as systemic anaphylaxis, milk allergy, or massive pulmonary migration of parasites.

f. Therapeutic plan. There is nothing specific that can be done to reverse the lung damage of affected cattle. Therefore, the main treatment goal is to reduce the adverse effects of the cell damage and inflammation as well as pulmonary edema.

(1) **Flunixin meglumine** at 2.2 mg/kg intravenously every 12 hours (extralabel use) is useful for its anti-inflammatory effects and furosemide at 0.4–1 mg/kg intravenously or intramuscularly every 12 hours reduces pulmonary edema.

(2) Equally important in management of these animals is the avoidance of stress or exercise.in hypoxic animals and removal of affected animals from any offending site, such as new pasture or feed source.

g. Prognosis. The prognosis is grave in severely affected animals, for which slaughter and salvage is often the most preferred option. For ARDS associated with 3-MI, clients should be cautioned that removal from pasture may not stop further clinical cases from developing in the short term.

h. Prevention. In ARDS associated with 3-MI, the herd should have limited access to pastures with pathogenic potential, such as alfalfa, kale, rape, turnips, or rapidly growing lush pasture. Where the problem is common and not readily avoidable by appropriate pasture management, monensin or lasalocid (200 mg/head/day) before and during pasture change can prevent the disease because these products inhibit overgrowth of ruminal lactobacilli that convert the dietary L-tryptophan to 3-MI.

5. Exercise-induced pulmonary hemorrhage **(EIPH)**

a. Patient profile and history. This disorder occurs in horses that undertake strenuous exercise such as racing. There is no relation of its occurrence to gender or finishing position, but its incidence may increase with age. The main complaint is either epistaxis at exercise or, more commonly, poor athletic performance without an obvious cause.

b. Clinical findings. In addition to epistaxis, clinical findings are likely to include problems related to athletic performance. Owners may report suspected EIPH horses as losing speed near the end of the race, after which they may take longer to "cool out." Affected horses may be observed to swallow more frequently during this cooling-off period.

c. Etiology and pathogenesis. The bleeding occurs in mainly the dorsal portions of the lung and appears to be related to events during strenuous exercise. Various theories have been offered to explain EIPH. Rupture of blood vessels is likely necessary for hemorrhage to occur.

(1) An older theory proposes that pulmonary hypertension and edema are the mechanisms of action. This was the rationale for treating the problems with furosemide. Furosemide therapy remains a popular treatment for "bleeders" on the race track, but there is no clinical evidence showing improvement of the lung bleeding. More recently, furosemide has been shown to have bronchodilating effects.

(2) A more recent theory is that EIPH occurs in horses that have subclinical lung disease, causing some degree of bronchiolar obstruction.

(a) This obstruction may be sufficient to prevent the filling of the alveoli distal to them when the respiratory rate is increased.

(b) Consequently, there is asynchrony between the air movement of the o b structed segment and the adjacent lung tissue. Because of the interdependence between different structures in the lung, this asynchrony may result in tearing of the lung parenchyma and resultant hemorrhage. This may be particularly true if there is lung scarring and pleural adhesions, which may result from past infections.

(3) This problem in race horses is the subject of study by many veterinary scientists because it appears to occur in a high proportion of race horses, and current understanding of the pathogenesis remains limited, which hampers effective treatment.

d. Diagnostic plan and laboratory tests

(1) Endoscopic examination of the trachea and collection of lower airway secretions for cytologic examination are the main methods of diagnosis. Confirmation of EIPH requires direct observation of blood in the trachea after a period of intense exercise. The former use of epistaxis as a diagnostic clinical sign is far too insensitive because whereas 75% of race horses have endoscopic evidence of hemorrhage in the trachea after the race, only 3% of these horses have blood present at the nostrils.

(2) Cytology of the lower airway secretions, either from transtracheal aspirates or bronchoalveolar lavage, shows alveolar macrophages packed with hemosiderin and, often, ingested red blood cells (hemosiderophages). An additional benefit of assessing lower ainvay cytology may be determining other underlying ainvay diseases that may be a predisposing factor in the genesis of EIPH. There appears to be no other test available for confirming the occurrence of EIPH.

e. Differential diagnoses. When EIPH is only suspected as a cause of performance problems in race horses, all other possible problems affecting performance, particularly lameness, should be ruled out. The differential diagnoses for epistaxis should include upper airway bleeding, such as guttural pouch mycosis, ethmoid hematomas, and trauma. In any horse with poor athletic performance, musculoskeletal, cardiac, and other respiratory problems must be ruled out before the EIPH is incriminated as the sole cause of the problem.

f. Therapeutic plan. There are no proven effective treatments for EIPH, partly because of the limited understanding of its pathogenesis. If a subclinical obstructive disease is present, then rest and environmental management are indicated.

(1) The most commonly used drug for treatment is furosemide at 0.3–0.8 mg/kg intravenously or intramuscularly 3 hours before racing, which is allowed in some racing jurisdictions for use in hones with EIPH confirmed by post-race endoscopy.

(2) Many other medications (although not approved for racing) are also in current use for treatment of this problem, but most lack any sound scientific basis for their use.

II. DISEASES OF THE THORAX

A. Diaphragmatic hernia

1. Patient profile and history. The type of animal affected with a diaphragmatic hernia varies. Often the history includes a previous trauma, such as dystocia, breeding, or foaling trauma, or severe physical exertion. Specific to cattle, traumatic reticuloperitonitis (TRP) is a main cause of this uncommon problem.

2. Clinical findings. Depending on the extent of the damage to the diaphragm and the amount of abdominal content herniated, the signs can range from mild colic and dyspnea to acute severe colic with tachypnea and obvious dyspnea.

a. Gastrointestinal sounds may be heard over the thorax, and lung sounds can be reduced or even absent over one side of the chest.

b. If the small intestine becomes strangulated in the hernia, there may be gastric reflux. In cattle with herniation associated with TRP, forestomach **stasis** can occur.

3. Diagnostic plan
   a. Radiography. When diaphragmatic hernia is suspected, chest radiography is usually diagnostic. Findings include loss of the diaphragmatic shadow and multiple fluid lines in the thorax that are associated with intestines in the thoracic cavity.
   b. Thoracocentesis generally yields a serosanguinous fluid, but this is in no way definitive for this specific diagnosis.
   c. **Electrocardiography.** Animals with diaphragmatic hernias usually have decreased amplitude of QRS complexes on an electrocardiogram (ECG).

4. Therapeutic plan and prognosis. Surgical closure of the diaphragmatic defect is the treatment of choice. Given the size of the large animal patients, the prognosis for success is only fair to guarded even if surgery is performed.

B. Pneumothorax

1. Patient profile and history. As in diaphragmatic hernia, all types of animals can be affected with pneumothorax. Usually there is some history of chest trauma. Undergoing an invasive technique, such as thoracocentesis or lung biopsy, may also be part of the recent history.

2. Clinical findings
   a. The main clinical sign with pneumothorax is a variable degree of dyspnea, the degree depending largely on the amount of air in the chest and subsequent lung collapse. If pneumothorax is associated with an open chest wound, this is usually obvious.
   b. On chest auscultation, there is an absence of lung sounds over the affected side because of collapse of lung parenchyma away from the chest wall. In some cases, the air also is found under the skin over the chest in the form of subcutaneous emphysema.

3. Etiology. Trauma to chest from external means, such as a penetrating wound, can result in pneumothorax. Alternatively, air leakage through the visceral pleura of the lung from lung damage, such as in rib fracture or ruptured emphysematous **bulla,** can also result in pneumothorax.

4. Diagnostic plan. Chest percussion often demonstrates a drum-like resonance with the presence of pneumothorax. Aspiration of air freely from the chest cavity by **thoracocentesis** confirms the diagnosis. With mild cases, chest radiographs may be the only diagnostic method, with **retraction** of the lung margins from the dorsal-most part of the thorax.

5. Therapeutic plan. The main goals in treatment of pneumothorax are to remove a sufficient amount of air from the chest cavity to resolve signs of dyspnea and to treat, if possible, the underlying cause.
   a. Any penetrating chest wound needs closure, at which time routine wound prophylaxis of antibiotics and administration of tetanus antitoxin (for horses) can be performed.
   b. To evacuate the chest of air, a teat canula can be placed into the pleural space and continuous suction applied. Medical **suction** devices are available for such purposes, but it is also possible to perform this in an ambulatory setting on dairy farms by using the suction from the milk line.

STUDY QUESTIONS

DIRECTIONS: Each of the numbered items or incomplete statements in this section is followed by answers or by completions of the statement. Select the ONE numbered answer or completion that is BEST in each case.

1. Which one of the following statements regarding Rhodococcus pneumonia is true?

(1) The treatment of choice is oral rifampin combined with a macrolide, such as lincomycin.
(2) Foals up to 4 months of age can be acutely infected from this soilborne organism; thus, a foal with clinical *R. equi* should be isolated from other foals to prevent the spread of infection.
(3) In addition to pulmonary abscessation, other sequelae to infection can include uveitis, nonseptic arthritis, ulcerative colitis, and vertebral body abscesses.
(4) This organism is able to live and grow within macrophages because it can prevent the fusion of phagosomes to lysosomes and has an antiphagocytic capsule (M protein) that prevents phagocytosis by granulocytes.
(5) Seroconversion to R. *equi,* in conjunction with signs of pneumonia and a leukocytosis, is the preferred method of diagnosis.

2. Which statement regarding enzootic pneumonia in calves is true?

(1) This disease is associated with bacterial pneumonia [secondary to bovine respiratory syncytial virus (BRSV) or parainfluenza-3 (PI-3) infection] or mycoplasmas, such as Mycoplasma mycoides.
(2) Enzootic pneumonia can occur in beef calves between ages 2 months and 5 months that are housed indoors in the fall and winter.
(3) Vaccination against viral agents such as BRSV or PI-3 in the first several weeks of life can help prevent this disease.
(4) Cold weather can precipitate this disease when producers leave housing ventilation open, exposing calves to cold outside air.
(5) The pathology is usually a consolidation of cranial, middle, and accessory lung lobes, with mycoplasmal involvement classically resulting in alveolitis and bronchiectasis.

3. Regarding pasteurellosis in ruminants, which one of the following statements is true?

(1) In sheep and goats, pasteurellosis due to *Pasteurella* haemolytica can occur in two forms: rapidly fatal septicemia with biotype T and pneumonia with biotype A.
(2) In cattle, shipping fever pneumonia due to pasteurellosis usually peaks several days after the stressful event.
(3) Vaccination against pasteurellosis in cattle should include the leukotoxin of P. haemolytica because it increases specific immunoglobulin A production in the lungs.
(4) Vaccination against pasteurellosis in cattle should include the respiratory viruses [infectious bovine rhinotracheitis (IBR), parainfluenza-3 (PI-3), bovine respiratory syncytial virus (BRSV)] because prior viral infection that accompanies the stress of handling is usually required for development of this disease.
(5) Pathogenic *P.* haemolytica strains can be part of normal pharyngeal flora in cattle, whereas the strains in sheep and goats spread from subclinical carrier animals.

4. Which one of the following statements regarding lungworms in large animals is true?

(1) The lungworm Dictyocaulus arnfieldi causes a patent infection in donkeys and results in a persistent cough in these infected animals.
(2) *Metastrongylus* species can cause lungworm in pigs raised outdoors, and though minimal clinical disease is found in experimental infection, the infection can result in exercise-induced cough and poor growth in field situations.
(3) Reinfection syndrome of D. *viviparus* in mature cattle causes disease because effective immunity is short-lived and has waned.
(4) *D. filaria* and Protostrongylusrufescens in sheep cause similar clinical signs, but because *P.* rufescens has a direct life cycle, it can cause more severe disease in adults.
(5) Although it causes a low-grade productive cough in growing kids and lambs, *Müellerius capillaris* can be effectively treated with a single dose of fenbendazole.

5. Which one of the following statements regarding pleural disease of large animals is true?

(1) Clinical signs of shallow breathing and chest pain are caused by the inflammation of pain receptors on the visceral pleural surfaces.
(2) Pleural fluid accumulation is usually readily apparent in both horses and cattle with pleuritis.
(3) Unilateral thoracic disease is possible in ruminants because of the intact mediastinum, but disease can spread bilaterally in horses and pigs because of the finer, often perforated mediastinum.
(4) Normal pleural fluid has a cell count of less than 10,000/μl white blood cells, but it is usually difficult to obtain in the clinically normal large animal.
(5) A foul odor of the pleural fluid in cases of pleuritis is suggestive of Escherichia coli growth, warranting inclusion of aminoglycoside treatment

6. A herd of cattle is moved from sparse, meager pasture to a lush grazing of turnip tops. The adult cows develop severe dyspnea, open-mouthed breathing, and in some, subcutaneous emphysema. Which one of the following statements best applies to this problem?

(1) The new pasture was likely higher in 3-methylindole (3-MI), which caused the direct lung damage by acting on the Clara cells.
(2) The lung damage from preformed toxin results in eventual hyaline membrane formation and irreversible fibrosis.
(3) Pretreatment of this herd with monensin or lasalocid before pasture change can prevent such outbreaks.
(4) Nursing calves are less severely affected, with signs of mild expiratory dyspnea and cough.
(5) Associated with the severe respiratory distress, lung sounds are typically harsh with crackles and wheezes over the entire lung field.

7. A horse shows blood at the nostrils following intense exercise. Which one of the following statements is true?

(1) The blood likely originated from the caudal lung lobes as a result of left heart failure and fluid overload of the lungs.
(2) The medication furosemide is allowable for this condition in certain racing jurisdictions.
(3) The blood most likely originated from the ethmoid region or guttural pouch because this is an uncommon clinical finding in race horses.
(4) The bleeding clearly indicates a performance-limiting problem.
(5) Treatment with procoagulants, such as aminocaproic acid or vitamin K_3, is indicated.

8. Which one of the following statements regarding thoracic disease in large animals is true?

(1) Diaphragmatic hernia in horses is most often caused by trauma, such as dystocia, whereas in cattle it has been linked to traumatic reticuloperitonitis.
(2) Signs of diaphragmatic hernia in horses can be mild to moderate colic and dyspnea, whereas cattle have occult herniation because the rumen is too large to herniate into the thorax.
(3) Gastrointestinal sounds (borborygmi) heard over the ventral chest of the hone are highly suggestive of diaphragmatic hernia.
(4) In cattle with pneumothorax, there is a drum-like resonance to percussion over the chest and very harsh lung sound ventrally because of the collapse of the lung.
(5) Sometimes uncovered incidentally, finding decreased amplitude of QRS and high spikes on the T wave of an electrocardiogram (ECG) of a horse are suggestive of diapragmatic hernia.

ANSWERS AND EXPLANATIONS

1. The answer is 3 [I A 2 b (2)]. The presently accepted and specific treatment for *Rhodococcus* equi pneumonia of foals is a combination of rifampin and erythromycin estolate. In horses, the use of oral lincomycin can induce fatal colitis. There is little benefit in isolating foals with R. equi pneumonia. Foals are resistant at 5 weeks of age; therefore, clinically affected foals pose little danger to most foals on the premises. The organism can prevent fusion of phagosome to lysosome but does not have an M protein capsule. Seroconversion is not a measure of disease because many foals seroconvert because of exposure to R. equi without ever developing R. equi pneumonia.

2. The answer is **2** [I A 3 a]. The disease can occur in any group of calves (not only dairy) that are between 2 months and 5 months of age and housed indoors, particularly when exposed to older cattle. Bacterial pneumonia, which is the end result of the enzootic pneumonia complex, may be secondary to infection by Mycoplasma species, but M. *mycoides* has not been described as a precursor agent. Prevention of enzootic pneumonia involves changing management practices. Vaccination has not proven beneficial because levels of colostral immunity interfere with the timing of vaccination. Cold outside air does not precipitate the disease. The buildup of noxious gases in poorly ventilated spaces has the negative impact on mucociliary clearance. The pathologic description in the last choice is accurate; however, this is the result of bacterial not mycoplasmal involvement.

3. The answer is **1** [I B 3 a]. Pasteurellosis in sheep and goats may appear as an acute septicemia as well as a primary pneumonia. The clinical signs of shipping fever in cattle may not occur until 2 weeks after the stressful event (most commonly co-mingling cattle). Viruses are not essential precursors for the disease. Vaccination is not known to produce protective levels of immunoglobulin A antibodies. Pathogenic Pasteurella *haemolytica* strains can be part of the normal pharyngeal flora in cattle, sheep, and goats.

4. The answer is 2 [I C 4 a]. *Dictyocaulus* arnfielddoes not cause clinical signs in its natural host, the donkey. Re-infection syndrome of *D. viviparus* is experienced in cattle with immunity (i.e., this is an immune-mediated reaction to re-infestation with massive numbers of infection larvae). *Protostrongylus rufescens* has an indirect life cycle, with the land snail as the intermediate host. *Müellarius capillaris* is difficult to treat, requiring treatment with fenbendazole over a 2-week period.

5. The answer is **4** [I E 5 c (3)]. The visceral pleura lacks pain receptors and is not the site of pain with pleuritis. In swine, the mediastinum is intact similar to ruminants. Pleuritis may not be obvious on clinical examination, particularly in cattle where the pleuritis is often more diffuse and a component of fibrinous pleuropneumonia. A necrotic foul odor to the breath or pleural fluid indicates the possibility of an anaerobic infection, suggesting treatment with metronidazole.

6. The answer is 3 [I F 4 a, b, h]. The clinical description best fits a diagnosis of acute respiratory distress syndrome in the bovine. In this condition, 3-methylindole (3-MI) is not ingested preformed but as dietary L-tryptophan. Clara cells metabolize the ruminally manufactured 3-MI to pneumotoxic metabolites, which injure the alveolar and capillary epithelium. Nursing calves (preruminants) are not affected because the source of L-tryptophan is the pasture. A very important clinical feature of this condition is the absence of breath sounds over the dorsal portions of the lung fields despite the obvious respiratory distress.

7. The answer is **2** [I F 5 b, f]. Blood at the nostrils following intense exercise of a horse is most likely exertion-induced pulmonary hemorrhage (EIPH). The present accepted theory of pathogenesis is that EIPH results from subclinical lung disease. Asynchrony in movement of various groups of alveoli in the dorsal (distal) airways results in the tearing of lung parenchyma and small vessel hemorrhage. This is a common condition in racehorses but has not been identified as performance limiting. Treatment with furosemide has shown the best clinical response.

8. The answer is **1** [II A 1]. Signs of diaphragmatic herniation in horses can be mild to severe colic. However, borborygmi over the ventral chest is not diagnostic of diaphragmatic herniation because the diaphragm is extremely concave (when viewed from the rear) in the horse, and normal abdominal sounds can be transmitted through the chest cavity. Although decreased QRS amplitude may be found in horses with diaphragmatic hernia, the high spike T wave is not a feature. In cattle with pneumothorax, there is a drum-like resonance but a complete absence of lung sounds over the affected chest.